## CASE REPORT

# Fulminant thrombosis of mechanical mitral valve prosthesis

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#### **Abstract**

In patients with prosthetic heart valves non-cardiac surgery may require temporary discontinuation of oral anticoagulation. Although the risk of valve related thromboembolic complications may generally be only slightly increased during the short perioperative period, in the presence of certain risk factors, replacement of oral anticoagulation with heparin is recommended. In the presented patient, unusually fulminant and finally fatal thrombosis of a mechanical mitral valve prosthesis developed within only 48 hours after non-cardiac surgery despite heparin treatment. The thrombosis was triggered by clinical conditions favouring a hypercoagulable state. This report dramatically shows that despite improvements in prosthetic heart valve design and in the management of anticoagulation, thrombosis remains one of the most dangerous complications after valve replacement with a mechanical prosthesis.

(Heart 2001;86:e16)

Keywords: complications; mitral valve; mechanical prosthesis; thrombosis

Thrombosis of a mechanical heart valve prosthesis is a potentially life threatening complication after valve replacement. Even with the use of oral anticoagulation the risk of thromboembolic complications averages between 1-2% per patient year<sup>1</sup> and depends on valve type, location, and adequacy of anticoagulation. The risk is increased in patients with a mitral valve prosthesis, atrial fibrillation, previous thromboembolic events, and hypercoagulable state.12 Especially in these patients, temporary discontinuation of oral anticoagulation for non-cardiac surgery confers additional risk and replacement with heparin is recommended.1 2 We report the case of a patient who developed unusually extensive thrombosis of mechanical mitral valve prosthesis despite heparin replacement treatment within only 48 hours after non-cardiac surgery in the presence of several factors favouring a hypercoagulable state.

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Accepted 25 July 2001

### Case report

A 60 year old man with chronic atrial fibrillation was initially admitted to our hospital with signs of cardiogenic and septic shock 48 hours after autologous skin graft for an extensive chronic venous ulcer of the leg. He had undergone mechanical tilting disc mitral valve replacement (Omnicarbon 31 M) for severe mitral valve stenosis five years previously. Preoperatively, local infection with Pseudomonas aeruginosa and Escherichia coli was treated for two weeks with ciprofloxacin 500 mg/day according to antibiotic sensitivity cultures. Antibiotic treatment was continued perioperatively. Preoperatively, no clinical signs of dysfunction of the mechanical mitral valve prosthesis were detected by the consulting cardiologist. Transthoracic echocardiography was without pathological findings. Ninety three hours before surgery, the patient's coumarin medication (international normalised ratio between 2.5 and 3.5 during the three preoperative months) was discontinued. Thirty four hours before surgery, his international normalised ratio was 1.4 and subcutaneous heparin (10<sup>4</sup> IU, three times daily) was started. Activated partial thromboplastin time was 68 seconds, 23 hours after initiation of heparin treatment. Heparin was stopped eight hours before surgery. On the second postoperative day, the patient's temperature rose to 40°C and he required intubation and mechanical ventilation for acute pulmonary oedema. On admission to our hospital, he presented with high fever (41°C), rapid atrial fibrillation, hypotension (80/40 mm Hg) despite intravenous catecholamine infusion, rales over both lungs, muffled valve sounds, and a holosystolic grade 1 murmur. No signs of emboli were encountered on peripheral vascular examination. A chest x ray confirmed diffuse interstitial pulmonary congestion. The patient's white blood cell count was  $16.9 \times 10^{9}$ /l, C reactive protein was 624.9 mg/dl, and haemoglobin was 11.2 g/dl (7 mmol/l). Clotting studies showed a platelet count of  $71.0 \times 10^9$ /l, antithrombin III of 34.2%, D dimers of 8.3 mg/dl (83 000 μg/l), and fibringen of 277 mg/dl (8.2 µmol/l), all consistent with disseminated intravascular coagulation. Blood cultures remained negative throughout his hospital stay. Mechanical valve prosthesis endocarditis was highly suspected by the referring physician.

2 of 3 Zielinska, Haegele, Firschke

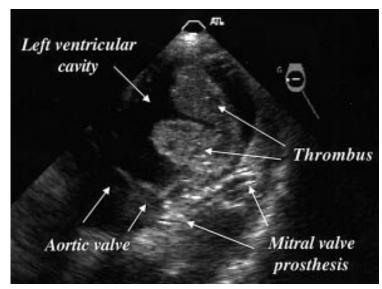


Figure 1 Transoesophageal echocardiogram showing huge masses attached to the left ventricular surface of the mitral valve prosthesis in the distal transgastric view as the multiplane probe is kept in the 0° position.

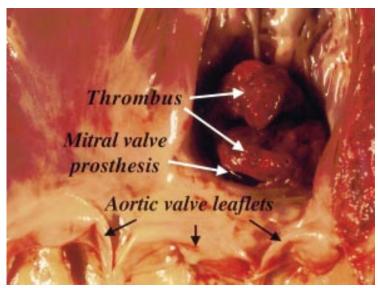


Figure 2 Postmortem examination found a large thrombus on the left ventricular side of the mitral valve prosthesis.

Transoesophageal echocardiography showed large and partly mobile masses of low echogenicity attached predominantly to the ventricular surface of the mitral tilting disc prosthesis (fig 1). Pressure half time on pulsed Doppler echocardiography was increased to 128 ms indicative of valvar obstruction. A mild eccentric transvalvar mitral regurgitation accompanied incomplete closure of the tilting disc.

Despite intensive treatment (catecholamine infusions, erythrocytes transfusions, antithrombin III substitution, broad spectrum antibiotic treatment, haemofiltration, continued mechanical ventilation) the patient remained hypotensive, anaemic, oliguric, acidotic, and unconscious. Six days after admission, he died of multiorgan failure.

Necropsy confirmed the presence of a large thrombus located mainly on the ventricular surface of the mitral valve prosthesis (fig 2) with obstruction of the valve orifice. No vegetations were found. Multifocal embolisation of mesenteric arteries, massive intestinal bleeding, and peritonitis were also seen. Diffuse cerebral oedema and local intracranial haemorrhage were found at necropsy. Additionally, a formerly unknown adenocarcinoma of the prostate (World Health Organization grade 2; Gleason score 5 (2 + 3); tumour, node, metastases classification apT1aN0M0) was discovered.

#### Discussion

The initial diagnosis was not unequivocal in our patient. Apparently sudden development of large masses of material attached to the prosthetic valve was indicative of thrombosis but interpretation of the valvar masses as vegetation, based on Duke criteria,<sup>3</sup> could not be ruled out. The appearance of the mass was unusual because of its dimensions and predominant location on the ventricular valve surface.

Although continuation of oral anticoagulation has been recommended during surgery on the skin,1 the decision to discontinue oral anticoagulation perioperatively in our patient was founded on the anticipation of major intraoperative bleeding caused by the extent of his leg disease. According to the American College of Cardiology/American Heart Association guidelines replacement with heparin was indicated because of the presence of a prosthetic valve in the mitral position plus atrial fibrillation as an additional risk factor for thrombotic complications. Management of heparin replacement treatment might have been improved in the present patient by the use of a loading bolus followed by intravenous infusion of heparin and repeated preoperative testing of activated partial thromboplastin time and dose adjustments. However, it appears unlikely that the present patient's course could have been prevented by these modifications. Massive thrombosis was most probably triggered by sepsis and subsequent disseminated intravascular coagulation.4 5 The presence of adenocarcinoma, although a potentially confounding factor for thrombosis through tissue factor production,6 was probably of minor relevance because the tumour was in a very early stage. The only treatment options for this patient were mitral valve replacement and thrombolysis, both with very high risk in this particular case. His poor general state was regarded as a contraindication for operation by the consulting cardiac surgeons. Thrombolytic treatment was not considered because the diagnosis of thrombus was ambiguous and intracranial and intestinal bleeding were suspected.

This report dramatically shows that, in the presence of certain trigger mechanisms, thrombus formation on a mechanical heart valve, a life threatening condition, may be very rapid and extensive despite anticoagulation.

Dr Zielinska was supported by grants from Dietmar-Zumpf Stiftung, Forstern-Preisendorf, Germany and DAAD, Bonn, Germany. We thank Jonathan Lindner MD and Donald Hall MD for thoughtful critique of the manuscript.

1 Bonow RO, Carabello B, de Leon AC Jr, et al. ACC/AHA guidelines for the management of patients with valvular

- heart disease: a report of the American College of Cardiology/American Heart Association task force on practice guidelines (committee on management of patients with valvular heart disease). *J. Am. Coll. Cardiol* 1998;32:1486–588.

  2 Gohlke-Barwolf C. Anticoagulation in valvar heart disease:
- 2 Goinge-Datwolf C. Anticoaguiaudri in valvar neart disease, new aspects and management during non-cardiac surgery. Heart 2000;84:567–72.
   3 Durack DT, Lukes AS, Bright DK. New criteria for diagnosis of infective endocarditis: utilisation of specific echocardiographic findings. Am J Med 1994;96:200–9.

- Esmon CT, Fukudome K, Mather T, et al. Inflammation, sepsis and coagulation. Haematologica 1999;84:254-9.
   Carey MJ, Rodgers GM. Disseminated intravascular coagulation: clinical and laboratory aspects. Am J Hematol 1998;59:65-73.
   Arkel YS. Thrombosis and cancer. Semin Oncol 2000;27: 362-74.
   Lengyel M, Fuster V, Keltai M, et al. Guidelines for management of left-sided prosthetic valve thrombosis: a role for thrombolytic therapy. J Am Coll Cardiol 1997;30: 1521-6. 1521-6.